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AUTHOR CORRECTION

Correction for Brady et al., "Poly(ADP-Ribose) Polymerases in Host-Pathogen Interactions, Inflammation, and Immunity"

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Page 7, paragraph 4: Lines 2 to 6 should read "... PARP12 shares some of the observed roles of PARP13 in RNA decay (36) and in the antiviral response. Like PARP13, it recognizes specific sequences in viral RNA and DNA (121, 122, 125, 137). Both proteins contain zinc finger domains in the N-terminal region. Recognition of RNA by these nucleic acid-binding domains is required for antiviral activity (129, 130, 137)."

Page 7, paragraph 6: Lines 1 to 7 should read "PARP7, -10, -12, and -13 are all induced by interferons, and all can inhibit viral replication (135, 136). PARP7 and PARP10, which are MARTs, are capable of translation inhibition. These proteins form complexes with ribosomes that are mediated by their N-terminal RNA-binding domains (137). Translation inhibition prevents viral growth by stopping viral protein synthesis. The finding that the induction of PARP7, -10, -12, and -13 can result in the inhibition of virus replication is consistent with previous results supporting the importance of PARPs in the inhibition of viral replication (135, 136)."

Page 9, paragraph 1: Lines 8 to 10 should read "... The RNA-binding PARP12 and -13 are found in stress granules, where PARPs are responsible for MARylation of various proteins such as the argonaute proteins, G3BP1, and TIA-1 (36)."

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